Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Circadian blood pressure adaptation and arterial stiffness in pregnancy: An exploratory study

Jose Ayala-Hernández

jrayalah77@gmail.com

Universidad Central de Venezuela https://orcid.org/0009-0008-3192-3939

Cristina López-Sánchez

Universidad Central de Venezuela

Steffany Castro

Universidad Central de Venezuela

Ana Barbosa

Universidad Central de Venezuela

Article

Keywords: Pregnancy, Gestational hypertension, Arterial stiffness, Ambulatory blood pressure monitoring, Circadian rhythm, Reproductive-aged women

Posted Date: March 12th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9088745/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: There is **NO** conflict of interest to disclose.

Abstract

Background

Maternal vascular adaptation influences blood pressure (BP) regulation and arterial stiffness. Ambulatory blood pressure monitoring (ABPM) allows calculation of the Ambulatory Arterial Stiffness Index (AASI), a non-invasive surrogate of vascular compliance. Data on these parameters in Latin American populations remain scarce.

Methods

This exploratory study included 81 women (36 nonpregnant; 45 healthy pregnant). 24-hour ABPM assessed BP, circadian patterns, and AASI. Comparisons were made by pregnancy status and gestational age (< 20 vs. ≥20 weeks) using Welch tests and bootstrap confidence intervals.

Results

Mean 24-hour BP was comparable between pregnant (111/66 mmHg) and nonpregnant women (115/69 mmHg; $p > 0.05$). AASI values were also similar between groups (0.33 ± 0.12 vs. 0.31 ± 0.18; $p > 0.05$), though pregnant women exhibited significantly reduced variability confirmed by bootstrap analysis. After 20 weeks, a significant shift from dipper to non-dipper systolic pattern occurred ($p < 0.00001$, Cramer's V = 0.703). Circadian patterns and AASI were not significantly associated.

Conclusions

Pregnancy involves distinct hemodynamic shifts, specifically a non-dipper pattern after mid-gestation and reduced AASI variability. These exploratory findings provide a physiological baseline for vascular adaptation in this population, informing future research on hypertensive disorders.

Summary Table

What is known about this topic
• Pregnancy involves significant hemodynamic adaptations and vascular remodeling to accommodate increased blood volume.
• The Ambulatory Arterial Stiffness Index (AASI) is an established surrogate marker of arterial stiffness and a predictor of cardiovascular risk in the general population.
• Evidence regarding AASI and circadian blood pressure patterns in Latin American pregnant cohorts is currently scarce.
What this study adds
• This exploratory study provides preliminary reference values for AASI and circadian profiles in a healthy Venezuelan obstetric population compared to non-pregnant controls.
• A significant shift from a "dipper" to a "non-dipper" systolic pattern was identified after 20 weeks of gestation, even in low-risk pregnancies.
• These findings suggest that 24-hour ABPM-derived indices like AASI can capture the physiological limits of vascular adaptation during healthy pregnancy.

1. Introduction

Pregnancy is a complex physiological process that involves profound cardiovascular and hemodynamic adaptations to ensure maternal and fetal well-being (1; 2). Among these changes, a decrease in mean arterial pressure is observed in midgestation, together with a reduction in systemic vascular resistance, largely mediated by estrogen and progesterone (3; 4).

Gestational hypertension remains a major public health concern due to its impact on maternal and perinatal morbidity and mortality, highlighting the need for diagnostic and predictive tools with greater sensitivity than isolated blood pressure measurements in the office (5).

Ambulatory blood pressure monitoring (ABPM) provides valuable information on circadian blood pressure patterns and allows calculation of additional parameters, such as pulse pressure and the Ambulatory Arterial Stiffness Index (AASI). AASI, derived from ABPM, is a reproducible surrogate of arterial stiffness. (6; 7; 8; 9; 10; 11; 12; 13; 14; 15).

In pregnant women, hormonal and hemodynamic changes can influence arterial stiffness and circadian blood pressure patterns, suggesting that AASI
could serve as a complementary tool for the early identification of women at risk of pregnancy hypertensive disorders (16; 17; 18).

Despite the clinical utility of the Ambulatory Arterial Stiffness Index (AASI), its reference values during normal vascular adaptation in Latin American pregnant populations remain unexplored. This study addresses this gap by characterizing circadian patterns and AASI in a Venezuelan cohort, providing essential data for regional cardiovascular health.

2. Methods

2.1. Study design and ethical considerations

We conducted an observational and analytical study using the database of the Research Group of the Chair of Clinical Medicine and Therapeutics A, School of Medicine, Luis Razetti, Central University of Venezuela. Data analyzed in this study were obtained from a previous investigation conducted by the same research group, which had been reviewed and approved by the Caracas Military Hospital Bioethics Committee (18). That study complied with the principles of the Declaration of Helsinki and the Declaration of Taipei. All participants provided written informed consent for the use of their clinical and ambulatory blood pressure monitoring data. For the present analysis, only deidentified data were used.

2.2. Population and sample

This exploratory pilot study was designed to characterize the distribution of AASI values in a specific regional cohort, providing a basis for future longitudinal investigations with larger sample sizes.

We included healthy women aged 18–45 with complete ABPM (70% valid readings, with 15-minute intervals during the day and 20-minute intervals during sleep); hypertension, diabetes, kidney disease, or antihypertensive therapy were exclusion criteria.

Participants were selected from a prospective database, and for this analysis, we included only women with no prior history of chronic hypertension, diabetes, or renal disease, as confirmed through medical record review at the time of ABPM placement, ensuring a true low-risk physiological profile.

2.3. Ambulatorybloodpressuremonitoring(ABPM)

Validated ABPM devices were used with standard daytime and nighttime intervals.

Circadian blood pressure patterns were classified as dipper, non-dipper, over-dipper, or riser, according to the nocturnal decrease in systolic blood pressure. The Ambulatory Arterial Stiffness Index (AASI) was defined using linear regression. For each participant, AASI was calculated as follows:

$$AASI = 1 - m(1)$$

where m is the slope of the ordinary least squares regression of diastolic
blood pressure (DBP, Y axis) on systolic blood pressure (SBP, X axis) across all 24-hour ABPM readings, with an unconstrained intercept.

2.4. Statistical analysis

Data were organized in Excel and processed with the Real Statistics Resource Pack. Categorical variables were summarized as frequencies and percentages, and continuous variables as mean ± standard deviation. Normality was tested with the Shapiro–Wilk. Group comparisons used ANOVA or

Student's t test, with chi-square/Fisher's exact for categorical data; effect size was estimated with Cramer's V. Variance homogeneity was assessed, and when heteroskedasticity was present, robust methods (Welch ANOVA/t test) were applied. Bootstrap resampling provided 95% confidence intervals for medians, and boxplots illustrated variability. A new AASI cutoff was proposed based on the 95% CI of mean values in healthy pregnancies. Statistical significance was set at $p < 0.05$.

3. Results

A total of 85 women were initially identified from the database. After excluding two cases with incomplete ABPM data, one patient with chronic hypertension and one with gestational diabetes, the final sample consisted of 81 women (Figure 1). Of these, 36 (44%) were non-pregnant women of reproductive age, and 45 (56%) were healthy pregnant women. The mean age was 32 ± 8.5 years in the non-pregnant group and 33 ± 8.6 years in the pregnant group.

Of the pregnant cohort, 23 (51%) women were evaluated before 20 weeks and 22 (49%) at or beyond 20 weeks of gestation. Mean 24-hour blood pressure was statistically comparable between pregnant and non-pregnant women (Table 1). AASI values were also similar between groups (0.33 ± 0.12 vs. 0.31 ± 0.18, respectively); although initial ANOVA suggested potential differences by gestational age, robust Welch tests did not confirm significance (Table 2, Figure 2). Notably, pregnant women exhibited narrower AASI variability, a finding further supported by bootstrap 95% confidence intervals (Table 3).

A definitive shift in circadian blood pressure architecture was identified based on gestational age. While the systolic dipper pattern predominated in both the reproductive-age group (86%) and the early pregnancy group (<20 weeks, 83%), a significant transition to a non-dipper profile was observed in pregnancies ≥20 weeks, where 91% of participants exhibited a non-dipper pattern (χ^2 $p < 0.00001$; Cramer's $V = 0.703$).

Table 1: Epidemiological and clinical data of patients evaluated through Ambulatory Blood Pressure Monitoring (ABPM)

Characteristics	Value	Student's t test p-value	Shapiro-Wilk p-value
Total (n)	81		-
Groups			
Reproductive age n (%)	36 (44)		0.777
Pregnant n (%)	45 (56)		0.201
1st trimester n (%)	11 (23)		
2nd trimester n (%)	19 (43)		
3rd trimester n (%)	15 (34)		
< 20 weeks n (%)	23 (51)		
≥ 20 weeks n (%)	22 (49)		
Age (years)			
Reproductive age (X±DE)	32±8.48	0.94	-
Pregnant (X±DE	33±8.64		
Systolic blood pressure			
Reproductive age (mmHg±DE)	115±12.06	0.162	-
Pregnant (mmHg±DE)	111±11.04		
Diastolic blood pressure			
Reproductive age (mmHg±DE)	69±9.93	0.065	-
Pregnant (mmHg±DE)	66±7.30		

This transition occurred despite mean 24-hour blood pressure levels remaining within normotensive ranges (111 ± 11 / 66 ± 7 mmHg), suggesting a systematic alteration in nocturnal blood pressure regulation rather than an onset of hypertension (Table 4, Figure 3). AASI did not vary significantly by circadian pattern or gestational subgroup (Table 5).

Table 2: Ambulatory Arterial Stiffness Index values in women of reproductive age and healthy pregnant women

Characteristics	AASI (X±DE)	Student's t test p-value	ANOVA p-value
Reproductive age	0.31±0.18	0.22	<0.01
Pregnant	0.33±0.12		
< 20 weeks	0.33±0.12	0.82†	0.027†
≥ 20 weeks	0.34±0.12	0.64‡	0.0486‡

Note: Tests of homogeneity of variances (Levene p = 0.045; Bartlett p = 0.020; Fisher's F-test p = 0.019) indicated heteroscedasticity between groups. Therefore, robust methods were applied: Welch ANOVA (p = 0.427) and Welch t-test (p = 0.446), which showed no significant differences in mean AASI values. The classical ANOVA and Student's t-test results are presented for transparency but should not be interpreted as a violation of variance assumptions.
† Compared with the reproductive-age group and <20 weeks of pregnancy
‡ Compared with the reproductive-age group and ≥20 weeks of pregnancy,
AASI: Ambulatory Arterial Stiffness Index.
Significance level p < 0.05.

Table 3: Median, interquartile range (IQR), and bootstrap confidence intervals (95% CI) of AASI in women of reproductive age and healthy pregnant women

Group	Median AASI	IQR (Q1–Q3)	Bootstrap 95% CI (Median)
Reproductive age	0.296	0.193 – 0.409	0.32 – 0.43
Pregnant	0.310	0.241 – 0.430	0.30 – 0.32

Note: Values are expressed as median (Q1–Q3). Bootstrap confidence intervals (95% CI) were estimated for medians.

Table 4: Circadian pattern of systolic blood pressure among women of reproductive age and healthy pregnant women

Circadian patterns SBP	Reproductive age (n)	Pregnants		χ^2 p-value	Cramer V
		< 20 weeks (n)	≥ 20 weeks (n)		
Dipper	31	19	2	< 0.0001	0.703
Nondipper	5	4	20		

SBP: Systolic Blood Pressure.
Significance level p < 0.05.

Using the conventional cut-off point of AASI ≥0.50, no significant associations were observed between circadian pattern and arterial stiffness in either group (Table 6). A new cut-off point of 0.37, derived from

the 95% confidence interval of the mean AASI in healthy pregnant women (0.30–0.37), was also tested. However, no significant associations were found when applying this threshold (Fisher's exact test, p = 0.1922) (Table 6).

In general, pregnant women showed more homogeneous AASI values with less dispersion compared to nonpregnant women, regardless of gestational age or circadian pattern. A consistent shift to a systolic non-dipper pattern was observed after 20 weeks of gestation.

Table 5: Circadian pattern of systolic blood pressure and AASI among women of reproductive age and healthy pregnant women

Circadian patterns	Reproductive age	Pregnants		p-value
SBP	X±DE	< 20 weeks X±DE	≥ 20 weeks X±DE	
Dipper	0.28±0.17	0.32±0.11		0.18612†
		0.33±0.11	0.27±0.03	0.771‡
Nondipper	0.44±0.16	0.34±0.13		0.18612†
		0.31±0.20	0.35±0.13	0.414‡

† p-value according to one-way ANOVA for the circadian pattern between healthy pregnant women and women of reproductive age.
‡ p-value according to Student's t-test for the circadian pattern between healthy pregnant women before and after 20 weeks of gestation.
SBP: Systolic Blood Pressure.
Significance level $p < 0.05$.

Table 6: Association between circadian pattern and AASI thresholds in reproductive-age and pregnant women

Groups	Dipper	Nondipper	Fisher's test p-value
Reproductive-age			
AASI≥0.5 (n)	3	3	0.0231
Pregnants			
AASI≥0.5 (n)	2	4	0.2634
AASI≥0.37(n)	5	8	0.1922

Values represent the number of individuals with AASI ≥0.50 and ≥0.37, including a proposed cutoff of 0.37 derived from the 95% CI of mean AASI in healthy pregnancies. Fisher's exact test was used to assess statistical significance between dipper and nondipper groups.
Significance level $p < 0.05$.

Discussion

Efforts to estimate cardiovascular risk in pregnant women, particularly through early and reliable methods to predict preeclampsia and thus prevent maternal-fetal complications, are increasingly emphasized (19).

The incidence of arterial hypertension during pregnancy has increased in recent years, in parallel with the obesity epidemic and the tendency to initiate pregnancy at more advanced maternal ages; both are considered predisposing factors. Hypertension in pregnancy not only produces maternal-fetal complications but may also represent the onset of future vascular and metabolic alterations. Moreover, it is not only the most frequent cause of maternal and infant morbidity and mortality, but it can also extend its negative influence on the long-term cardiovascular health of the offspring (20).

Therefore, pregnancy can be considered a "stress test" for the future cardiovascular health of the woman, since it requires cardiovascular adaptations that may have long-term consequences when accompanied by a hypertensive disorder (21;22).

ABPM for 24 hours is currently recommended for hypertensive pregnant women or those with risk factors (6; 15). The Ambulatory Arterial Stiffness Index (AASI) has been defined in the general population as an independent cardiovascular risk marker capable of predicting cardiovascular mortality, cerebrovascular events, and target organ damage, including renal failure, which frequently affects women in the puerperium. AASI is highly useful and can be calculated with relative ease from a standard ABPM study.

Our study included two comparable groups of healthy women: those of reproductive age and pregnant women, further subdivided by gestational age (<20 and ≥20 weeks). Both groups shared similar clinical–epidemiological characteristics and mean age.

According to current ESC guidelines, their ambulatory blood pressure values remained within the range considered non-elevated, confirming that the study population can be considered normotensive. This provides a reliable baseline for exploring differences in arterial stiffness and circadian patterns as physiological adaptations of pregnancy (15).

Regarding AASI, both groups showed comparable mean values, but their variability differed. Women of reproductive age showed greater dispersion, while pregnant women exhibited more homogeneous values, consistent with physiological stabilization during gestation.

During pregnancy, hemodynamic adaptations occur, including changes in circadian blood pressure patterns, presumably influenced by trophoblastic invasion and hormonal changes that optimize microcirculation, endothelial function, and arterial distensibility (4). Park et al. (2022) demonstrated that hormonal influences can modify arterial stiffness in young women, which may explain the variability observed in women of reproductive-age in our study. In contrast, pregnancy-related vascular adaptations appear to stabilize arterial stiffness, resulting in a more consistent hemodynamic behavior (23). However, in pregnant women, vascular adaptations can stabilize arterial stiffness, resulting in more consistent hemodynamic behavior.

Different studies have reported contradictory results regarding AASI in various populations. Although arterial stiffness correlates with methods such as pulse wave velocity (PWV), there is an ongoing debate as to whether AASI truly reflects stiffness as a parameter and how it should be interpreted (24). Flórez-Rios et al. evaluated arterial stiffness using PWV and the aortic augmentation index (AIx) as predictors of low birth weight but found no significant differences in stiffness values between pregnant women with and without low-birth-weight newborns. These results suggest that arterial stiffness may not change significantly during pregnancy or that different cut-off values may be required in this population (25).

Given that pregnant women are a more susceptible risk group, refinement of AASI as a predictive tool for the evaluation of cardiovascular risk is highly relevant. However, there is limited evidence on the use of ABPM in studies composed exclusively of women. Some investigations, such as those by Mortazavi et al. (2023), who demonstrated the impact of sleep blood pressure classification on nocturnal nondipping, and Hermida et al. (2003), included predominantly female populations and allowed subgroup analyses. These studies analyzed pulse pressure and other parameters of ABPM, but few have specifically evaluated AASI during pregnancy (26; 27).

In our study, circadian systolic blood pressure patterns were determined in both groups. Among women of reproductive age, the systolic dipper pattern predominated, as expected in a low-risk population

(28). However, in pregnant women, the results differed by gestational age.

The most striking finding of this study is the robust shift toward a systolic non-dipper pattern after the 20th week of gestation. Unlike the general population, where a non-dipper status is often associated with increased cardiovascular risk and target organ damage, in healthy pregnancy, this phenomenon appears to represent a pivotal physiological adaptation. We hypothesize that this loss of nocturnal dipping serves to maintain stable and continuous placental perfusion during sleep, compensating for the systemic vasodilation and increased blood volume characteristic of mid-to-late gestation. The high effect size (Cramer's V = 0.703) underscores that this is not a marginal observation but a consistent feature of vascular remodeling in our cohort.

Although previous reports have described circadian changes during pregnancy, our results demonstrate a consistent and statistically robust shift in this cohort. This adaptation may reflect the need to maintain stable placental perfusion and highlights the value of ambulatory blood pressure monitoring as a complementary tool to understanding vascular adaptation during pregnancy, while future studies should clarify its role in risk assessment.

In previous research from our group, Zambrano and López (18) demonstrated that pregnant women shift from a systolic dipper pattern in the first half of pregnancy (<20 weeks) to a systolic nondipper pattern after 20 weeks, probably as a physiological vascular adaptation. This finding was reproduced in the present study, with a strong association between the circadian pattern and gestational age.

When analyzing the association between the circadian pattern and AASI, our results did not show significant differences in pregnant women, suggesting that vascular behavior during gestation remains relatively uniform regardless of the circadian pattern. Previous studies reported heterogeneous associations between AASI and circadian patterns, with differences mainly in extreme cases (risers, over-dippers); this variability is consistent with reports from Jerrard-Dunne, Raimondo, Boos, and more recent analyses (Ayala-Hernández) (16; 24; 29; 30).

Beyond the similarity of mean AASI values, the key finding of this study lies in the reduced variability observed in pregnant women. This homogeneity, confirmed by narrower interquartile ranges and bootstrap confidence intervals, suggests vascular stabilization during gestation. This consistency may reflect physiological adaptations that optimize placental perfusion and protect against extreme circadian blood pressure patterns. Importantly, the application of robust statistical methods (Welch tests and bootstrap resampling) strengthens the reliability of these conclusions. Although the exploratory threshold of 0.37 did not discriminate circadian patterns in healthy pregnancies, it may serve as a reference band for future studies in hypertensive or high-risk cohorts.

In contrast, pregnant women did not show an intragroup association between AASI and the circadian pattern, likely due to physiological
adaptations to pregnancy. Future studies should evaluate AASI in pregnant women with extreme circadian patterns (over-dippers, risers), chronic hypertension, preeclampsia, or high cardiovascular risk,

ideally with multivariate adjustments for confounders such as body mass index, maternal age, parity, prior preeclampsia, and diabetes. In the general population, AASI ≥0.5 is linked to organ damage, but pregnancy may require lower cutoffs (11; 12).

We acknowledge that the sample size is modest. However, as an exploratory study, its primary value lies in the rigorous application of 24-hour ABPM and the first-time reporting of AASI in this specific regional population. To ensure the reliability of our inferences despite the sample size and the observed heteroscedasticity, we employed robust statistical methods, including Welch's tests and bootstrap resampling for confidence intervals. These findings reveal a clear and statistically robust trend ($p < 0.00001$, Cramer's $V = 0.703$) toward a non-dipper systolic pattern after 20 weeks of gestation. Such preliminary data provide an essential baseline for future longitudinal investigations into the physiological limits of vascular adaptation during healthy pregnancy.

Maintaining this line of research is essential. Our observations should be applied to subgroups of pregnant women with hypertensive disorders, given the demonstrated utility of arterial stiffness (measured by PWV and pulse wave reflection) as a predictor of preeclampsia, in combination with uterine artery Doppler and angiogenic biomarkers (31).

Conclusion

This study provides foundational evidence on the physiological limits of vascular adaptation in a healthy Latin American obstetric cohort. Our findings demonstrate that while mean ambulatory blood pressure remains within normotensive ranges, pregnancy induces a significant and systematic shift toward a non-dipper systolic pattern after the 20th week of gestation. This transition likely represents a necessary hemodynamic adjustment to ensure stable nocturnal placental perfusion.

Furthermore, the Ambulatory Arterial Stiffness Index (AASI) exhibited a notable reduction in variability compared to non-pregnant controls, suggesting a state of vascular stabilization unique to healthy gestation. Although AASI did not show a direct association with circadian patterns in this normotensive group, the establishment of these preliminary reference values is a critical step toward identifying early vascular maladaptation.

Ultimately, 24-hour ABPM-derived indices, particularly the circadian profile and AASI, offer a more granular understanding of maternal hemodynamics than isolated office measurements. These tools hold promise for the early identification of women at risk for hypertensive disorders, and their integration into clinical monitoring—especially after mid-gestation—warrants further validation in high-risk populations.

6. Future Work

Future research should evaluate the Ambulatory Arterial Stiffness Index (AASI) in high-risk pregnancies, including women with chronic hypertension, preeclampsia, or extreme circadian patterns, to determine its utility as a reliable marker in these subgroups. It will also be important to test lower cutoff points

adapted to pregnancy physiology and to integrate AASI with complementary tools such as uterine artery Doppler—assessing resistance indices, flow velocity, and persistence of the protodiastolic notch—together with angiogenic biomarkers, aiming for a more comprehensive assessment of vascular adaptation and cardiovascular risk during gestation.

Declarations

Author Contributions: C.L-S. and J.A-H. conceived and designed the study. S.C. and A.B. served as research assistants and were responsible for data acquisition and database management. J.A-H. performed the statistical analysis and drafted the manuscript. All authors reviewed and approved the final version.

Patient Consent Statement

In the original study, all participants gave their informed written consent. Given that this study is a retrospective analysis of the anonymized database, the requirement for additional patient consent was fulfilled in the original protocol.

Permission to Reproduce Material from Other Sources

Not applicable, as no material from external sources was reproduced in this study.

Clinical Trial Registration

Not applicable, as this is an observational study based solely on pre-existing data.

Conflicts of Interest

The authors declare that they have no competing interests.

Data Availability Statement

The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the authors used Grammarly Version 1.146.3.0 to assist with language refinement. After using this tool, the authors reviewed and edited the content as needed and assume full responsibility for the content of the published article.

References

1. J. Duvekot, E. Cheriex, F. Pieters, P. Menheere, L. Peeters, Early pregnancy changes in hemodynamics and volume homeostasis are consecutive adjustments triggered by a primary fall in systemic vascular tone, American Journal of Obstetrics and Gynecology 169 (6) (1993) 1382– 1392. doi:10.1016/0002-9378(93)90405-8.
2. J. Duvekot, L. Peeters, Maternal cardiovascular hemodynamic adaptation to pregnancy, Obstetrical & Gynecological Survey 49 (12 Suppl) (1994) S1–14. doi:10.1097/00006254-199412011-00001.
3. R. Christiason, Studies on blood pressure during pregnancy: Influence of parity and age, American Journal of Obstetrics and Gynecology 125 (1976).
4. G. Osol, N. L. Ko, M. Mandalà, Plasticity of the maternal vasculature during pregnancy, Annual Review of Physiology 81 (2019) 89–111. doi:10.1146/annurev-physiol-020518-114435.
5. World Health Organization, Información general sobre la hipertensión en el mundo. Día Mundial de la Salud, World Health Organization, Geneva (2023).
6. D. Stisman, M. Bendersky, P. Cufaro, P. Rodríguez, et al., Mediciones ambulatorias de la presión arterial, Sociedad Argentina de Hipertensión Arterial.Documento de consenso del Grupo de Trabajo de Mediciones Ambulatorias de Presión Arterial y Telemedicina (2020). URL https://saha.org.ar/uploads/pdf/LibroMapa-SahaSep2020.pdf
7. A. Haider, M. Larson, S. Franklin, D. Levy, Systolic blood pressure, diastolic blood pressure, and pulse pressure as predictors of risk for congestive heart failure in the Framingham Heart Study, Annals of Internal Medicine 138 (1) (2003) 10–16. doi:10.7326/0003-4819-138-1-200301070-00006.
8. M. Safar, H. Boudier, Vascular development, pulse pressure, and the mechanisms of hypertension, Hypertension 46 (1) (2005) 205–209. doi:10.1161/01.HYP.0000167992.80876.26.
9. M. Valero, J. Caputo, G. Guzmán, S. Graf, R. Armentano, Simulación de la influencia de la elasticidad parietal sobre el índice ambulatorio de rigidez arterial (aasi), Revista Ingeniería Biomédica 3 (6) (2009) 18–25.
10. A. Benetos, C. Adamopoulos, J. Bureau, M. Temmar, C. Labat, K. Bean, et al., Determinants of accelerated progression of arterial stiffness in normotensive subjects and in treated hypertensive subjects over a 6-year period, Circulation 105 (10) (2002) 1202–1207. doi:10.1161/hc1002.105135.
11. C. Mathers, D. Loncar, Updated projections of global mortality and burden of disease, 2002-2030: data sources, methods and results, World Health Organization (2005).
12. S. Laurent, P. Boutouyrie, R. Asmar, I. Gautier, B. Laloux, L. Guize, et al., Aortic stiffness is an independent predictor of all-cause and cardio-vascular mortality in hypertensive patients, Hypertension 37 (5) (2001) 1236–1241. doi:10.1161/01.hyp.37.5.1236.
13. S. Laurent, J. Cockcroft, L. Van Bortel, P. Boutouyrie, C. Giannattasio, D. Hayoz, et al., Expert consensus document on arterial stiffness: methodological issues and clinical applications, European Heart Journal 27 (21) (2006) 2588–2605. doi:10.1093/eurheartj/ehl254.
14. M. Kikuya, J. Staessen, T. Ohkubo, L. Thijs, H. Metoki, K. Asayama, et al., Ambulatory arterial stiffness index and 24-hour ambulatory pulse pressure as predictors of mortality in Ohasama, Japan, Stroke 38 (4) (2007) 1161–1166. doi: 10.1161/01.STR.0000259604.67283.69.

15. J. W. McEvoy, C. P. McCarthy, R. M. Bruno, S. Brouwers, M. D. Canavan, C. Ceconi, et al., 2024 ESC Guidelines for the management of elevated blood pressure and hypertension, European Heart Journal 45 (38) (2024) 3912–4026. doi:10.1093/eurheartj/ehae178. URL https://academic.oup.com/eurheartj/article/45/38/3912/7741010
16. J. R. Ayala-Hernández, C. López-Sánchez, O. M. Ayala-Hernández, C. E. Palencia-Tejedor, Ambulatory arterial stiffness index: Regression method comparison and its association with pulse pressure and circadian patterns, The Journal of Clinical Hypertension 27 (12) (2025) e70191, open Access. doi:10.1111/jch.70191.
17. M. Said, R. Eppinga, E. Lipsic, N. Verweij, P. van der Harst, Relationship of arterial stiffness index and pulse pressure with cardiovascular disease and mortality, Journal of the American Heart Association 7 (2) (2018). doi:10.1161/JAHA.117.007621.
18. S. G. Zambrano Zabala, C. López-Sánchez, Variabilidad diurna y nocturna de la presión arterial en embarazadas sanas y mujeres en edad reproductiva a través del monitoreo ambulatorio de presión arterial, Medicina Interna (Caracas) 26 (1) (2010) 28–37, tab., graf. URL https://pesquisa.bvsalud.org/portal/resource/pt/lil-772227
19. B. Álvarez Álvarez, N. Martell-Claros, M. Abad-Cardiel, J. García-Donaire, Hipertensión en el embarazo: riesgos vasculares, Hipertensión y Riesgo Vascular 34 (2) (2017) 85–92. doi:10.1016/j.hipert.2016.06.002.
20. J. Hutcheon, S. Lisonkova, K. Joseph, Epidemiology of preeclampsia and the other hypertensive disorders of pregnancy, Best Practice & Research Clinical Obstetrics & Gynaecology 25 (4) (2011) 391–403.
21. L. Young, L. Cho, Unique cardiovascular risk factors in women, Heart 105 (21) (2019) 1656–1660. doi:10.1136/heartjnl-2018-314268.
22. S. J. Zieman, V. Melenovsky, D. A. Kass, Mechanisms, patho-physiology, and therapy of arterial stiffness, Arteriosclerosis, Thrombosis, and Vascular Biology 25 (5) (2005) 932–943. doi:10.1161/01.ATV.0000160548.78317.29. URL http://dx.doi.org/10.1161/01.ATV.0000160548.78317.29
23. J.-S. Park, J.-H. Shin, J.-B. Park, D.-J. Choi, H.-J. Youn, C.-G. Park, et al., Central hemodynamics and the discrepancy between central blood pressure and brachial blood pressure, Medicine (Baltimore) 101 (40) (2022) e30484. doi:10.1097/MD.0000000000030484. URL http://dx.doi.org/10.1097/MD.0000000000030484
24. D. Di Raimondo, A. Casuccio, R. Di Liberti, G. Musiari, V. Zappulla, A. D'Angelo, et al., Ambulatory arterial stiffness index (AASI) is unable to estimate arterial stiffness of hypertensive subjects: Role of nocturnal dipping of blood pressure, Current Hypertension Reviews 13 (2) (2017) 121–131. doi:10.2174/1573402113666170621110305. URL http://dx.doi.org/10.2174/1573402113666170621110305
25. A. M. Flórez-Ríos, J. Alzate-Giraldo, J. J. Zuleta-Tobón, L. I. Gallego-Vélez, et al., Variables hemodinámicas de la rigidez arterial en embarazadas y su relación con bajo peso para la edad

gestacional, Ginecología y Obstetricia de México 89 (1) (2021) 34–42. doi:10.24245/gom.v89i1.4471. URL https://doi.org/10.24245/gom.v89i1.4471

26. R. C. Hermida, D. E. Ayala, J. R. Fernández, A. Mojón, I. Alonso, M. F. Aguilar, M. Iglesias, Variación circadiana de la presión arterial en gestantes sanas, con hipertensión gestacional y con preeclampsia, Medicina Clínica 120 (14) (2003) 521–528. doi:10.1016/S0025-7753(03)73764-6. URL https://doi.org/10.1016/S0025-7753(03)73764-6
27. B. J. Mortazavi, J. L. Martinez-Brockman, B. Tessier-Sherman, M. Burg, M. Miller, Z. Nowroozilarki, et al., Classification of blood pressure during sleep impacts designation of nocturnal nondipping, PLOS Digital Health 2 (6) (2023) e0000267. doi:10.1371/journal.pdig.0000267. URL http://dx.doi.org/10.1371/journal.pdig.0000267
28. G. Parati, G. Stergiou, E. O'Brien, R. Asmar, L. Beilin, G. Bilo, et al., European society of hypertension practice guidelines for ambulatory blood pressure monitoring, Journal of Hypertension 32 (2014) 1359– 1366. doi:10.1097/HJH.0000000000000221
29. P. Jerrard-Dunne, A. Mahmud, J. Feely, Circadian blood pressure variation: relationship between dipper status and measures of arterial stiffness, Journal of Hypertension 25 (6) (2007) 1233–1239. doi:10.1097/HJH.0b013e3280eec79f. URL http://dx.doi.org/10.1097/HJH.0b013e3280eec79f
30. C. J. Boos, L.-T. Toon, H. Almahdi, The relationship between ambulatory arterial stiffness, inflammation, blood pressure dipping and cardiovascular outcomes, BMC Cardiovascular Disorders 21 (1) (2021) 139. doi:10.1186/s12872-021-01946-2. URL http://dx.doi.org/10.1186/s12872-021-01946-2
31. K. Phan, Y. H. Gomez, J. Gorgui, A. El-Messidi, R. Gagnon, H. A. Abenhaim, et al., Arterial stiffness for the early prediction of pre-eclampsia compared with blood pressure, uterine artery doppler and angiogenic biomarkers: a prospective cohort study, BJOG: An International Journal of Obstetrics & Gynaecology 130 (8) (2023) 932–940. doi:10.1111/1471-0528.17430. URL http://dx.doi.org/10.1111/1471-0528.174

Figures

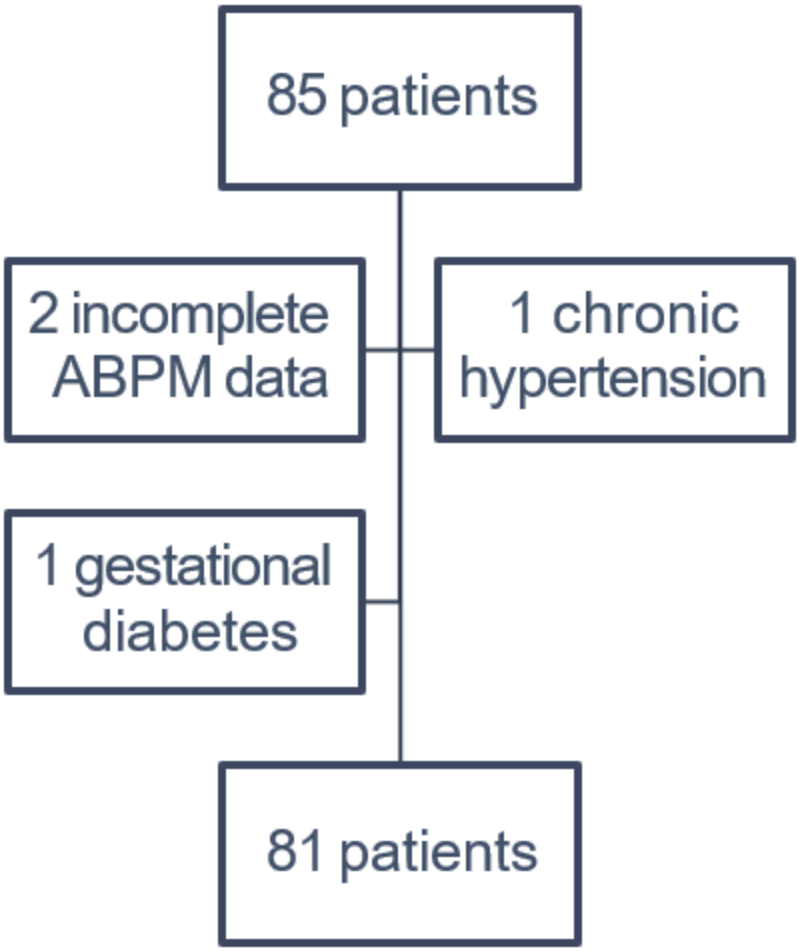


Figure 1

Patient selection (sample)

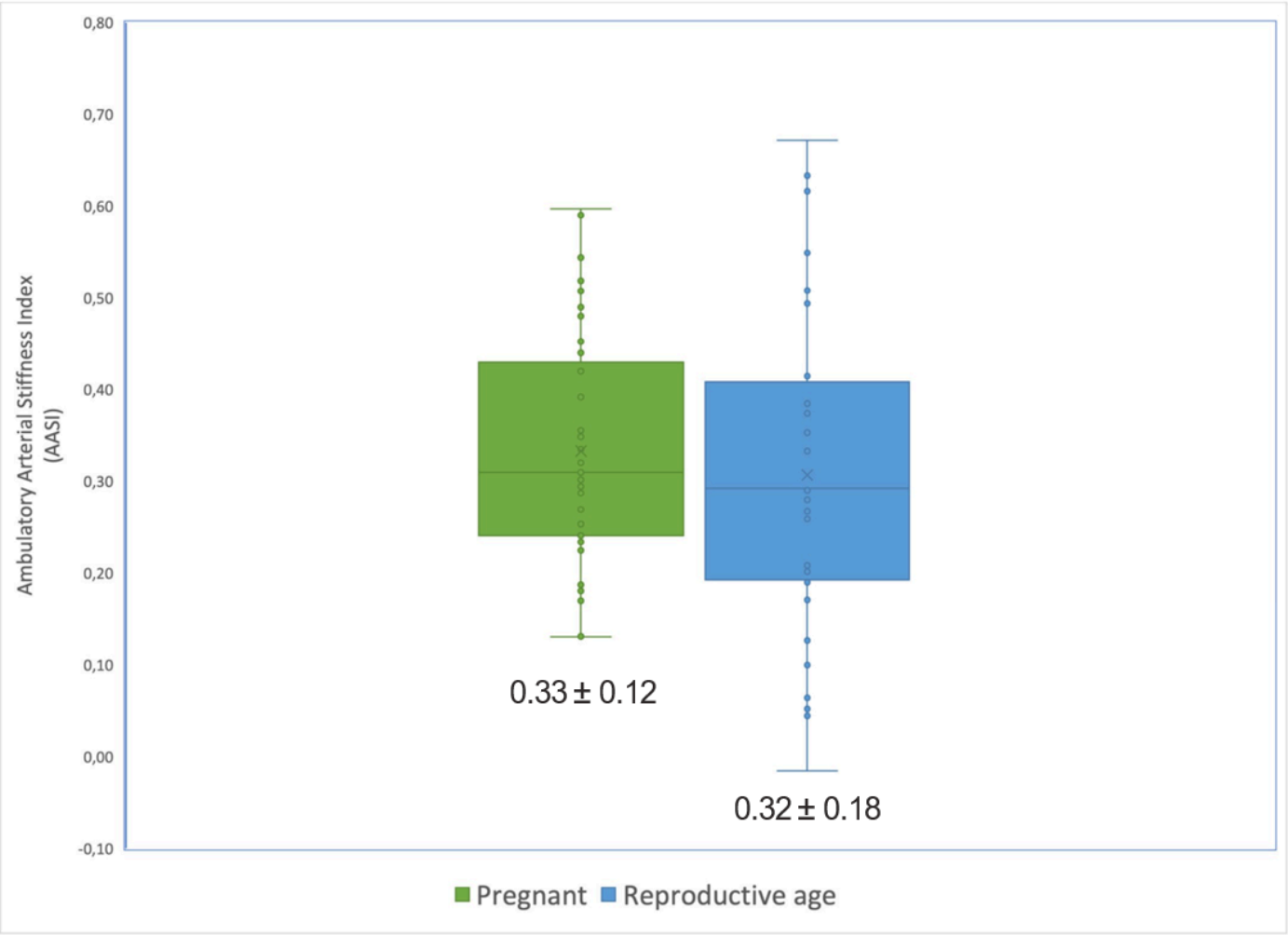


Figure 2

Ambulatory Arterial Stiffness Index in healthy pregnant women and women of reproductive age.

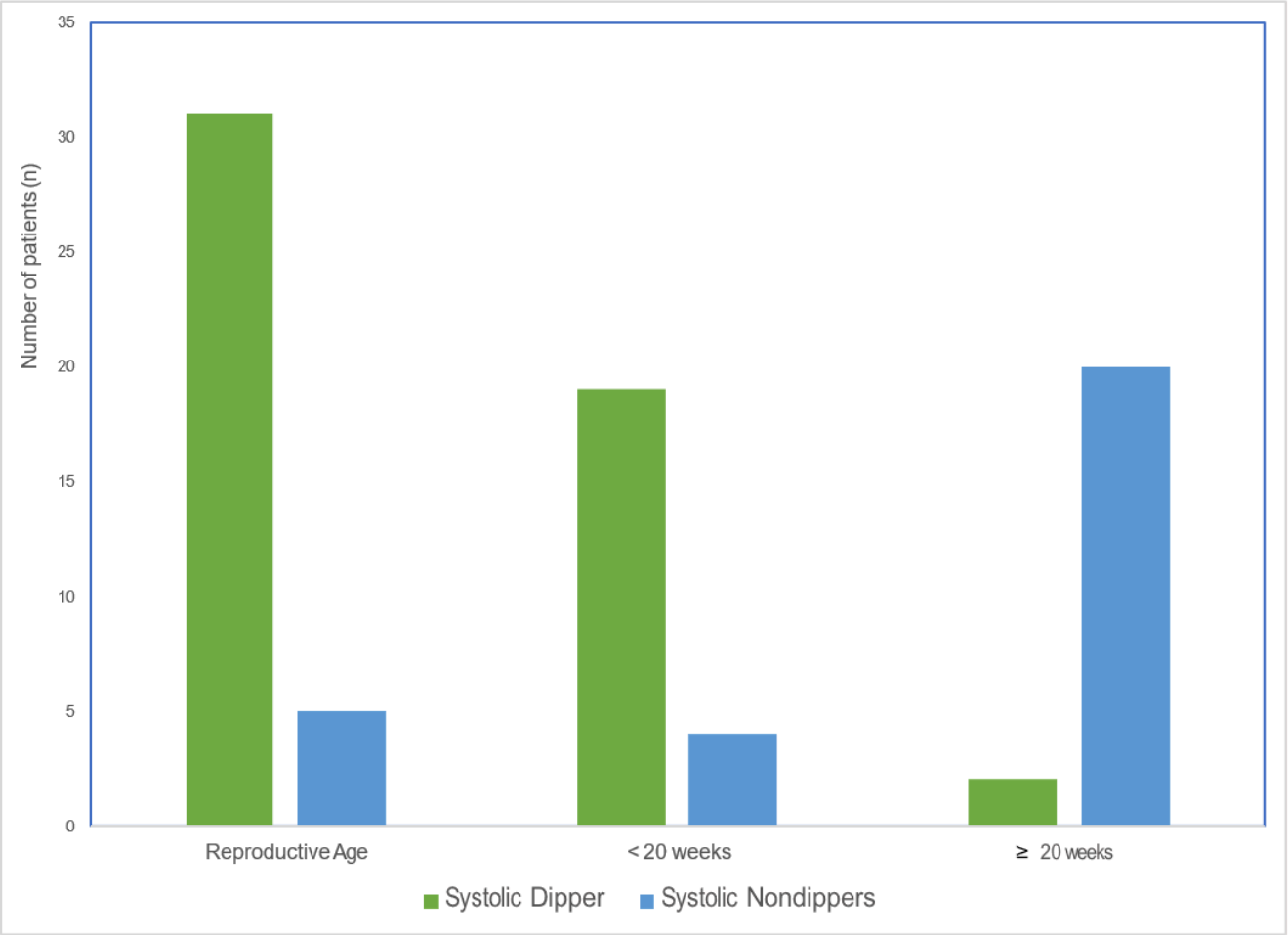


Figure 3

Circadian pattern among women of reproductive age and pregnant women before and after the 20th week of gestation